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10/533,115	04/29/2005	Yoshiko Takayama	2005_0740A	2341

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WENDEROTH, LIND & PONACK, L.L.P.
1030 15th Street, N.W.,
Suite 400 East
Washington, DC 20005-1503

EXAMINER	
WANG, CHANG YU	

ART UNIT	PAPER NUMBER
1649	

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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/533,115	TAKAYAMA ET AL.	
	Examiner	Art Unit	
	CHANG-YU WANG	1649	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *13 October 2009*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *14* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *14* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

RESPONSE TO AMENDMENT

Status of Application/Amendments/claims

1. Applicant's amendment filed 10/13/09 is acknowledged. Claims 1-13 and 15-22 are cancelled. Claim 14 is amended. Claim 14 is pending in this application and under examination in this office action.

2. Any objection or rejection of record, which is not expressly repeated in this office action has been overcome by Applicant's response.

3. Applicant's arguments filed on 10/13/09 have been fully considered but they are not deemed to be persuasive for the reasons set forth below.

Claim Rejections/Objections Withdrawn

4. The rejection of claims 13-16 and 18-21 under 35 U.S.C. 102 (b) as being anticipated by Nordisk (WO98/58646, published on Dec 30, 1998 as in IDS) is withdrawn in response to Applicant's amendment to the claims and cancellation of claims 13, 15-16 and 18-21.

Claim Rejections/Objections Maintained

In view of the amendment filed on 10/13/09, the following rejections are maintained.

Claim Rejections - 35 USC § 103

5. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

The factual inquiries set forth in *Graham* **v.** *John Deere Co.*, 383 U.S. 1, 148 USPQ 459 (1966), that are applied for establishing a background for determining obviousness under 35 U.S.C. 103(a) are summarized as follows:

1. Determining the scope and contents of the prior art.
2. Ascertaining the differences between the prior art and the claims at issue.
3. Resolving the level of ordinary skill in the pertinent art.
4. Considering objective evidence present in the application indicating obviousness or nonobviousness.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

Claim 14 is rejected under 35 U.S.C. 103(a) as being unpatentable over Nordisk (WO98/58646, published on Dec 30, 1998 as in IDS) in view of Perez-Santonja et al. (Am J. Ophthalmol. 1999. 127:497-504, cited previously), WO98/44922 (Yang et al, published Oct 15, 1998, as in IDS) and WO97/43278 (Ankersen et al, published Nov 20, 1997, as in IDS) as evidenced by Suzuki et al. (see p. 550, abstract, Suzuki et al. Curr

Eye Res. 2000. 21:550-553, cited previously) and Fini et al. (see p. S12 2nd col., Fini et al. Arch Dermatol. Res. 1998. 290: S12-S23, cited previously) and the data of cornea (p.3-4, retrieved from the NEI website, www.nei.nih.gov/health/cornealdisease, cited previously). Note that WO98/44922 and WO97/43278 are newly cited references, which are necessitated by Applicant's amendment to the claims.

Claim 14 as amended is drawn to a method of recovering decreased corneal sensitivity after surgery in a subject with a damaged or cut corneal nerve axon, which comprises topically administering an effective amount of t-butyl 6-amino-2-(3-(1H-indol-3-yl)-2-((4-(2-oxo-2,3-dihydrobenzoimidazol-1-yl)piperidine-1-carbonyl)amino)propionylamino)hexanoate or 1-(3-(N-(5-bromopyridin-2-yl)-N-(3,4-dichlorobenzyl)amino)propyl)-3-(3-(1H-imidazol-4-yl)propyl)thiourea to the eye of a subject with a damaged or cut corneal nerve axon in need of the recovery of corneal sensitivity.

Nordisk (WO'646) teaches a method of treating several eye diseases including glaucoma, inflammation of corneal stroma, stromal keratitis, iritis, retinitis, cataract and conjunctivitis by somatostatin (i.e. a somtatostatin agonist for SSTR2 and SSTR4; see abstract; p.3, p.5; p9-23 for somatostatin receptor agonists). The limitation of recovering corneal sensitivity recited in instant claim 14 is an inherent result of administration of somatostatin in these diseases because these diseases cause dry eye and corneal sensitivity; and the structure of cornea is within the eye and eye contains conjunctiva, cornea, iris, retina and optic nerve (see p.2 of the data retrieved from the NEI website, www.nei.nih.gov/health/cornealdisease, cited previously). Thus, the effects of

somatostatin receptor agonists on recovering corneal sensitivity and dry eye would inherently occur upon administration of somatostatin receptor agonists into the eye. In addition, conjunctivitis, inflammation of corneal stroma, stromal keratitis also result in corneal epithelium defect as evidenced by Suzuki et al. (see p. 550, abstract, Suzuki et al. Curr Eye Res. 2000. 21:550-553, cited previously) and Fini et al. (see p. S12 2nd col., Fini et al. Arch Dermatol. Res. 1998. 290: S12-S23, cited prevously) because these diseases also affect corneal epithelium. Particularly, conjunctivitis, inflammation of corneal stroma and stromal keratitis (herpes virus infection on cornea) would also cause corneal epithelium defect because these illness would affect cornea and the cornea encompasses five layers of cells including epithelium, Bowman's membrane, stroma, Descemet's membrane and endothelium (from outside to inside of the cornea; see p.3-4 of the data retrieved from the NEI website, www.nei.nih.gov/health/cornealdisease, cited previously). Suzuki et al. teach that conjunctival inflammation (conjunctivitis) induces migration of Langerhans cells to corneal to attach corneal epithelium and consequently affect corneal allografts (see p. 550, abstract). Fini et al. teach that corneal ulceration can be caused by stromal keratitis (herpes virus infection on cornea) and stromal ulceration is initiated by defective healing of corneal epithelium (see p.S13, 2nd col & table 1).Thus, the treatment of conjunctivitis, inflammation of corneal stroma, stromal keratitis by somatostatin as disclosed by Nordisk (WO'646) would inherently treat corneal epithelium defect. Since the materials (somatostatin receptor agonists), affected tissue and area within the patients suffering from eye diseases are the same between the claimed method and the method of Nordisk (WO'646), the administration of

somatostatin receptor agonists to the eye would inherently have the effects on the entire eye structures including corneal nerve axon and corneal epithelium.

But Nordisk does not teach that the decreased corneal sensitivity occurs after surgery and does not teach t-butyl 6-amino-2-(3-(1H-indol-3-yl)-2-((4-(2-oxo-2,3-dihydrobenzoimidazol-1-yl)piperidine-1- carbonyl)amino)propionylamino)hexanoate as a somatostatin receptor SSTR2 agonist or 1-(3-(N-(5-bromopyridin-2-yl)-N-(3,4-dichlorobenzyl)amino)propyl)-3-(3-(1H-imidazol-4-yl)propyl)thiourea as a somatostatin receptor SSTR4 agonist as recited in the instant claim.

Perez-Santoja et al. teach that laser in situ keratomileusis to correct myopia or photorefractive keratectomy can decrease corneal sensitivity (see abstract & p. 497, col.2, in particular). The teaching of Perez-Santoja et al. supports that an eye surgery such as laser in situ Keratomileusis to correct myopia or photorefractive keratectomy also causes decreased corneal sensitivity as other eye diseases taught by Nordisk.

WO98/44922 teaches t-butyl 6-amino-2-(3-(1H-indol-3-yl)-2-((4-(2-oxo-2,3-dihydrobenzoimidazol-1-yl)piperidine-1- carbonyl)amino)propionylamino)hexanoate as an SSTR2 agonist (see abstract; p.2, p.8, p. 13-15, in particular). WO97/43278 teaches 1-(3-(N-(5-bromopyridin-2-yl)-N-(3,4-dichlorobenzyl)amino)propyl)-3-(3-(1H-imidazol-4-yl)propyl)thiourea as an SSTR4 agonist (see abstract; p.2-22, in particular).

It would have been obvious to one of ordinary skill in the art at the time the instant invention was made to use somatostatin or a somtatostatin agonist for SSTR2/SSTR4 to treat or recover decreased corneal sensitivity in a subject with a damaged or cut corneal nerve axon wherein the decreased corneal sensitivity results

from surgery. The person of ordinary skill in the art would have been motivated to do so with an expectation of success because an eye surgery can cause decreased corneal sensitivity and somatostatin, a somtatostatin agonist for SSTR2/SSTR4, has successfully been used to treat or recover decreased corneal sensitivity in patients with a damaged corneal nerve axon, such as patients with glaucoma, conjunctivitis, inflammation of corneal stroma, stromal keratitis, which are disorders with a damaged corneal nerve and defective corneal epithelium as taught by Nordisk. Thus, the results of treating or recovering decreased corneal sensitivity after surgery using somatostatin or a somtatostatin agonist for SSTR2/SSTR4 would have been expected.

In addition, it would have been obvious to a skilled artisan at the time the instant invention was made to use a SSTR2 agonist such as t-butyl 6-amino-2-(3-(1H-indol-3-yl)-2-((4-(2-oxo-2,3-dihydrobenzoimidazol-1-yl)piperidine-1-carbonyl)amino)propionylamino)hexanoate or a SSTR4 agonist such as 1-(3-(N-(5-bromopyridin-2-yl)-N-(3,4-dichlorobenzyl)amino)propyl)-3-(3-(1H-imidazol-4-yl)propyl)thiourea to recover decreased corneal sensitivity in a subject with a damaged or cut corneal nerve axon wherein the decreased corneal sensitivity results from surgery. The person of ordinary skill in the art would have been motivated to do so with an expectation of success because an eye surgery can cause decreased corneal sensitivity as taught by Perez-Santoja and somatostatin, a SSTR2 or SSTR4 somatostatin agonist, has successfully been used to recover decreased corneal sensitivity in patients with a damaged corneal nerve axon, such as patients with glaucoma, conjunctivitis, inflammation of corneal stroma, stromal keratitis, which are

disorders with a damaged corneal nerve and defective corneal epithelium as taught by Nordisk. Thus, the results of recovering decreased corneal sensitivity after surgery using t-butyl 6-amino-2-(3-(1H-indol-3-yl)-2-((4-(2-oxo-2,3-dihydrobenzoimidazol-1-yl)piperidine-1- carbonyl)amino)propionylamino)hexanoate or 1-(3-(N-(5-bromopyridin-2-yl)-N-(3,4-dichlorobenzyl)amino)propyl)-3-(3-(1H-imidazol-4-yl)propyl)thiourea would have been expected because t-butyl 6-amino-2-(3-(1H-indol-3-yl)-2-((4-(2-oxo-2,3-dihydrobenzoimidazol-1-yl)piperidine-1- carbonyl)amino)propionylamino)hexanoate is an SSTR2 agonist and 1-(3-(N-(5-bromopyridin-2-yl)-N-(3,4-dichlorobenzyl)amino)propyl)-3-(3-(1H-imidazol-4-yl)propyl)thiourea is an SSTR4 agonist as taught by WO98/44922 and WO97/43278 respectively.

On p. 4-5 of the response, Applicant argues that Nordisk is not aware that Somatostatin agonist promotes extension of corneal nerve axon because optic nerves are different from trigerminal nerves and cited Attachment A in support of the argument. Applicant argues that Perez-Santoja only teaches that LASIK or PRK decreases corneal sensitivity but does not teach Somatostatin agonist promotes extension of corneal nerve axon. Applicant argues that it is impermissible hindsight to combine the recited references and a skilled artisan would not have been motivated to combine the cited references, and thus would not have an expectation of success in obtaining the claimed invention. Applicant further argues that the cited references do not teach that use of t-butyl 6-amino-2-(3-(1H-indol-3-yl)-2-((4-(2-oxo-2,3-dihydrobenzoimidazol-1-yl)piperidine-1- carbonyl)amino)propionylamino)hexanoate or 1-(3-(N-(5-bromopyridin-

2-yl)-N-(3,4-dichlorobenzyl)amino)propyl)-3-(3-(1H-imidazol-4-yl)propyl)thiourea in the claimed method has an unpredictable effect on corneal nerve axon extension. Applicant's arguments have been fully considered but they are not persuasive.

In response to applicant's arguments against the references individually, one cannot show nonobviousness by attacking references individually where the rejections are based on combinations of references. See *In re Keller*, 642 F.2d 413, 208 USPQ 871 (CCPA 1981); *In re Merck & Co.*, 800 F.2d 1091, 231 USPQ 375 (Fed. Cir. 1986).

In response to applicant's argument that the examiner's conclusion of obviousness is based upon improper hindsight reasoning, it must be recognized that any judgment on obviousness is in a sense necessarily a reconstruction based upon hindsight reasoning. But so long as it takes into account only knowledge which was within the level of ordinary skill at the time the claimed invention was made, and does not include knowledge gleaned only from the applicant's disclosure, such a reconstruction is proper. See *In re McLaughlin*, 443 F.2d 1392, 170 USPQ 209 (CCPA 1971).

In addition, in considering the disclosure of a reference, it is proper to take into account not only specific teaching of the reference but also the inferences which one skilled in the art would be reasonably be expected to drawn therefrom. *In re Preda*, 401 F.2d 825, 159 USPQ 342, 344 (CCPA 1968).

In response to applicant's argument that there is no motivation to combine the cited references, it is not necessary that the claimed invention be expressly suggested

in any one or all of the references to justify combining their teachings; rather the test is what the combined teachings of the references would have suggested to those of ordinary skill in the art. *In re Keller*, 642 F.2d 413, 208 USPQ 871 (CCPA 1981).

The motivation to combine can arise from the expectation that the prior art elements will perform their expected functions to achieve their expected results when combined for their common known purpose. MPEP. §2144.07. Specific statements in the references themselves which would spell out the claimed invention are not necessary to show obviousness, since questions of obviousness involve not only what references expressly teach, but what they would collectively suggest to one of ordinary skill in the art. See *CTS Corp. v. Electro Materials Corp. of America* 202 USPQ 22 (DC SNY 1979); and *In re Burckel* 201 USPQ 67 (CCPA 1979).

In this case, Nordisk teaches a method of treating several eye diseases including glaucoma, inflammation of corneal stroma, stromal keratitis, iritis, retinitis, cataract and conjunctivitis by somatostatin (i.e. as it relates to claims 13-16; see abstract; p.3, p.5; p9-23 for somatostatin receptor agonists). Although Nordisk does not specifically teach a subject with a damaged or cut corneal nerve axon, as previously made of record, it is known in the art that glaucoma also causes corneal/optic nerve damage as evidenced by the data of NEI (retrieved on April 9, 2009 from the website of http://www.nei.nih.gov/glaucoma/printpage.asp?ref=http://www.nei.nih.gov/glaucoma/content/english/faq.asp, cited previously). Thus, patients with glaucoma meet the limitation of a subject with a damaged or cut corneal nerve axon. In addition, as previously made of record, the diseases taught by Nordisk cause dry eye and corneal sensitivity; the eye

contains conjunctiva, cornea, iris, retina and optic nerve, and the structure of cornea is within the eye. Note that the claim does not recite trigeminal or optic nerves. Regardless of what mechanisms underlying the somatostatin (an SSTR2 or SSTR4 agonist) action are (i.e. whether or not it promotes corneal nerve extension), as long as somatostatin (an SSTR2 or SSTR4 agonist) can treat glaucoma and other eye diseases, the effect on the damaged or cut corneal nerve axon in glaucoma is intrinsic and thus would recover the decreased corneal sensitivity in the patients. Note that

"The discovery of a previously unappreciated property of a prior art composition, or of a scientific explanation for the prior art's functioning, does not render the old composition patentably new to the discoverer." *Atlas Powder Co. v. Ireco Inc.*, 190 F.3d 1342,1347, 51 USPQ2d 1943, 1947 (Fed. Cir. 1999). Thus the claiming of a new use, new function or unknown property which is inherently present in the prior art does not necessarily make the claim patentable. *In re Best*, 562 F.2d 1252, 1254, 195 USPQ 430, 433 (CCPA 1977). " See MPEP § 2112.01 [R-3].

In addition, although Nordisk does not teach that the decreased corneal sensitivity occurs after surgery as recited in instant claim 14, Perez-Santoja teaches that laser in situ keratomileusis to correct myopia or photorefractive keratectomy can decrease corneal sensitivity (see p. 497, abstract & col.2). Note that laser in situ keratomileusis and photorefractive keratectomy are a surgery that causes decreased corneal sensitivity. Thus, it would have been obvious to a skilled artisan at the time the instant invention was made to use somatostatin, a SSTR2 or SSTR4 somatostatin agonist to recover decreased corneal sensitivity in a subject with a damaged or cut corneal nerve axon wherein the decreased corneal sensitivity results from surgery. The person of ordinary skill in the art would have been motivated to do so with an expectation of success because an eye surgery can cause decreased corneal sensitivity and somatostatin, a SSTR2 or SSTR4 somatostatin agonist has successfully

been used to recover decreased corneal sensitivity in patients with a damaged corneal nerve axon, such as patients with glaucoma, conjunctivitis, inflammation of corneal stroma, stromal keratitis, which are disorders with a damaged corneal nerve and defective corneal epithelium as taught by Nordisk. Thus, the results of recovering decreased corneal sensitivity after surgery using somatostatin, a SSTR2 or SSTR4 somatostatin agonist would have been expected.

Furthermore, although Nordisk and Perez-Santoja do not specifically teach that t-butyl 6-amino-2-(3-(1H-indol-3-yl)-2-((4-(2-oxo-2,3-dihydrobenzoimidazol-1-yl)piperidine-1- carbonyl)amino)propionylamino)hexanoate or 1-(3-(N-(5-bromopyridin-2-yl)-N-(3,4-dichlorobenzyl)amino)propyl)-3-(3-(1H-imidazol-4-yl)propyl)thiourea as an SSTR2 and SSTR4 agonist respectively, WO98/44922 teach t-butyl 6-amino-2-(3-(1H-indol-3-yl)-2-((4-(2-oxo-2,3-dihydrobenzoimidazol-1-yl)piperidine-1-carbonyl)amino)propionylamino)hexanoate as an SSTR2 agonist (see abstract; p.2, p.8, p. 13-15, in particular), and WO97/43278 teaches 1-(3-(N-(5-bromopyridin-2-yl)-N-(3,4-dichlorobenzyl)amino)propyl)-3-(3-(1H-imidazol-4-yl)propyl)thiourea as an SSTR4 agonist (see abstract; p.2-22, in particular). Thus, it would have been obvious to a skilled artisan at the time the instant invention was made to use t-butyl 6-amino-2-(3-(1H-indol-3-yl)-2-((4-(2-oxo-2,3-dihydrobenzoimidazol-1-yl)piperidine-1-carbonyl)amino)propionylamino)hexanoate or 1-(3-(N-(5-bromopyridin-2-yl)-N-(3,4-dichlorobenzyl)amino)propyl)-3-(3-(1H-imidazol-4-yl)propyl)thiourea to recover decreased corneal sensitivity in a subject with a damaged or cut corneal nerve axon wherein the decreased corneal sensitivity results from surgery. The person of ordinary

skill in the art would have been motivated to do so with an expectation of success because an eye surgery can cause decreased corneal sensitivity, and somatostatin, a SSTR2 or SSTR4 somatostatin agonist has successfully been used to recover decreased corneal sensitivity in patients with a damaged corneal nerve axon, such as patients with glaucoma, conjunctivitis, inflammation of corneal stroma, stromal keratitis, which are disorders with a damaged corneal nerve and defective corneal epithelium as taught by Nordisk. Thus, the results of recovering decreased corneal sensitivity after surgery using t-butyl 6-amino-2-(3-(1H-indol-3-yl)-2-((4-(2-oxo-2,3-dihydrobenzoimidazol-1-yl)piperidine-1- carbonyl)amino)propionylamino)hexanoate or 1-(3-(N-(5-bromopyridin-2-yl)-N-(3,4-dichlorobenzyl)amino)propyl)-3-(3-(1H-imidazol-4-yl)propyl)thiourea would have been expected because t-butyl 6-amino-2-(3-(1H-indol-3-yl)-2-((4-(2-oxo-2,3-dihydrobenzoimidazol-1-yl)piperidine-1-carbonyl)amino)propionylamino)hexanoate is an SSTR2 agonist and 1-(3-(N-(5-bromopyridin-2-yl)-N-(3,4-dichlorobenzyl)amino)propyl)-3-(3-(1H-imidazol-4-yl)propyl)thiourea is an SSTR4 agonist as taught by WO98/44922 and WO97/43278 respectively.

Conclusion

6. NO CLAIM IS ALLOWED.

7. Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

8. Any inquiry of a general nature or relating to the status of this general application should be directed to the Group receptionist whose telephone number is (571) 272-1600.

Papers relating to this application may be submitted to Technology Center 1600, Group 1649 by facsimile transmission. The faxing of such papers must conform with the notice published in the Official Gazette, 1096 OG 30 (November 15, 1989). Should applicant wish to FAX a response, the current FAX number for Group 1600 is (571) 273-8300.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Chang-Yu Wang whose telephone number is (571) 272-4521. The examiner can normally be reached on Monday-Thursday from 8:30 AM to 6:30 PM. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Jeffrey Stucker, can be reached at (571) 272-0911.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

/CYW/
Chang-Yu Wang, Ph.D.
January 7, 2010

/Christine J Saoud/
Primary Examiner, Art Unit 1647